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July 27, 2007

TO THE ADDRESSEES LISTED ON
SCHEDULE A ATTACHED HERETO

RE: Christian Holinka v A. W. Chesterton, et al

Ladies and Gentlemen:

You have asked me to express an opinion regarding the role, if any, of each defendant listed in Schedule A, in causation of the medical condition in the above referenced litigation.

The following information has been provided by your office for our review.

1. Plaintiff's Answers to Interrogatories
2. Social Security Records
3. Complaint
4. Deposition Under Oral Examination of Christian Holinka, dated 02/12/07, Vol. 1
5. Deposition Under Oral Examination of Christian Holinka, dated 02/22/07, Vol. 2
6. Deposition Under Oral Examination of Christian Holinka, dated 03/01/07, Vol. 3
7. Medical Records and Reports Including Those of Drs.: B. Dsouza, H. Hibshoosh, K. Kodsi, L. Lund, R. Meyers, J. Molina, J. Sonett, A. Stancato-Pasik, J. Strauchen, R. Taub
8. Medical records of: St. Luke's - Roosevelt Hospital Center, Columbia Thoracic, Columbia Presbyterian Eastside Radiology, Columbia University Medical Center

In August 2006, at age 69, Dr. Christian Holinka presented with dyspnea, large right pleural effusion, and multiple right pleural based lesions. Thoracoscopy and right pleural biopsy provided the diagnosis of malignant mesothelioma, biphasic type. (Lund, Strauchen reports) Dr. Holinka was treated with intrathoracic chemotherapy with gamma-interferon and cisplatin for 3 cycles and systemic chemotherapy with Alimta and Cisplatin for 3 cycles. His current condition is not reported.

Radiological evaluations demonstrated large right pleural effusion with multiple pleural based lesions and a clear left lung. (Dsouza, Kodsi, Stancato-Pasik reports)

Lung tissue was not present to assess for interstitial fibrosis or ferruginous bodies. (Strauchen report)

There was no significant past medical history provided.

Christian Holinka was born in Bad Altheide, Poland in 1937 where he lived until 1950 when he moved to Olpe, West Germany. (dep pg 15) In 1956, at the age of 19 he moved to the United States and (dep pg 22-23) entered the US Army, training as a medical laboratory technician. (dep pg 27) During his two months of training, he used a variety of laboratory equipment including Bunsen burners that utilized asbestos pads which he handled. (dep pg 27) Following his training he was station at the 98th General Hospital in Neubruecke, Germany until 1959. (dep pg 32) Dr. Holinka worked in all branches of the clinical medical laboratory including bacteriology, biochemistry, hematology, and pathology. (dep pg 32-33) He regularly handled Bunsen burner pads, which he estimated replacing once a week, an activity that took seconds. (dep pg 35) He frequently

used mittens to shield himself from hot glass work (dep pg 38). He left the military in 1959 and returned to the United States, eventually residing in California and attending the University of California, Berkeley. (dep pg 44) Dr. Holinka studied physiology and spent time in laboratories where he handled asbestos mittens and Bunsen burner pads. (dep pg 80-81) He moved to New York City in 1971 attending graduate school at Columbia University and the State University of New York – Stony Brook until 1974 when he returned to California. From 1974 to 1977 he was a post doctoral student at the University of Southern California performing biological research. During his education process, Dr. Holinka repeatedly described exposure to Bunsen burner pads and mittens. From 1977 to 1989 Dr. Holinka was an instructor and then assistant professor in obstetrics, gynecology and reproductive science. His principal duties were research. (dep pg 149-150) He continued to report exposure to Bunsen burner pads and mittens (dep pg 151- 162), and possibly autoclaves. (dep pg 236) He left academia in 1989 and worked for 3 years at Organon as the director of reproductive medicine without any known association with asbestos. (dep pg 169) From 1992 to 1996 he was the assistant director at Johnson & Johnson Pharmaceuticals Research Institute, then from 1996 to 1997 he worked for Kyowa Hakko Kogio Pharmaceutical and in 1998 he began his career as a consultant, all without known asbestos association. (dep pg 170, 171, 173-175)

As to the laboratory equipment Dr. Holinka identified as asbestos containing (Bunsen burner pads and mittens), he repeatedly states in his testimony that he cannot specifically identify a manufacturer of either the Bunsen burner pads or the mittens he utilized.

It is now claimed that Dr. Holinka developed mesothelioma, that asbestos exposure caused his mesothelioma, and that Bunsen burner pads and mittens contributed to risk of the mesothelioma.

That Dr. Holinka developed mesothelioma is correct. The diagnosis was reached based upon disease presentation and progression, and appropriate radiologic, pathologic, and immunohistochemical findings.

That the mesothelioma was caused by exposure to asbestos has not been established. There is a lack of clinicopathologic evidence of an asbestos etiology, lack of a history of asbestos exposure that could be considered causal, and the presence of a scientifically plausible alternative etiology.

1. Lack of clinicopathologic evidence of an asbestos etiology. There are no clinicopathologic findings to indicate either asbestos exposure or risk.

    a. Radiologic: Radiologic evaluations do not demonstrate parenchymal abnormalities or pleural plaques, the most sensitive radiologic markers of past asbestos exposure (ACR, Nishimura). It is quite clearly understood that absence of such radiologic observations does not exclude attribution of risk to asbestos. (Roggli 2002) However, radiologic findings that would establish asbestos exposure are absent.

    b. Pathologic: Determination of lung tissue fibrosis, ferruginous (asbestos) bodies, or uncoated asbestos fiber burden was not reported. Such evaluation could determine whether the mesothelioma is asbestos related (Battifora, Craighead 1988, Hasan, Roggli 1992, Thurlbeck) and has been termed crucial in litigation. (McCaughey 1985)

2. Lack of a history of asbestos exposure that could be considered causal: Information is provided in testimony on very limited association with asbestos containing materials. The history is neither substantive nor compelling as to direct exposure appropriate to development of mesothelioma risk, as certainly would trades of insulator or shipyard worker. (Helsinki 1997)

    The occupational history, as described in testimony, cannot be rationally considered as establishing risk of asbestos related disease, including mesothelioma.

3. The presence of a scientifically plausible alternative etiology: A mesothelioma need not be asbestos related. The causal relationship between asbestos exposure and mesothelioma, as in all malignancies, is not absolute. Where there is insufficient evidence of causative asbestos exposure, then a mesothelioma can be appropriately considered idiopathic, without an established cause. It is well accepted that there is an incidence of mesothelioma that is not due to asbestos exposure but due to spontaneous tumor formation. (Churg and Green, Craighead 1995, Ilgren, Peterson, Spirtas) An authoritative and well referenced textbook states:

> In our own studies, approximately 11% of mesotheliomas have a lung asbestos content indistinguishable from background, and perhaps 10 to 20% of cases are not the result of asbestos exposure. The size of the exposed population at risk for mesothelioma and the relative rarity of the disorder suggest variable individual susceptibilities, possibly genetically mediated. The observation that a substantial proportion of patients with malignant mesothelioma have no identifiable exposure to asbestos has led investigators to look for other potential etiologic or predisposing factors.
>
> *Roggli VL, Spom TA, Mesothelioma. Ch 5, In: Pathology of Asbestos-Associated Disease, 2nd Ed, (Roggli VL, Oury TD, Spom TA, eds), Springer: New York, 2004, pg 108.*

Established etiologic criteria have not been met in this case, and there is a scientifically reasonable alternative diagnosis. This mesothelioma cannot be considered asbestos related unless and until lung tissue pathologic evaluation establishes asbestos exposure causation.

However, if the mesothelioma in this case is erroneously deemed to be asbestos related, the assertion that Bunsen burner pads or mittens created, or contributed to, mesothelioma risk in this case is not correct. There is an inherent lack of contribution to dose and risk, and an inability of the asbestos fiber type possibly used in Bunsen burner pads and mittens to create mesothelioma risk.

1. Inherent lack of contribution to dose and risk by Bunsen burner pads and mittens. It is considered extremely unlikely that such products could have contributed to risk in this case when dose generation potential is considered. The two major factors to consider in dose generation are intensity (concentration) and time. Dose is the product of concentrations (f/cc) over the time, or duration, of exposures (years). It is expressed as fibers per cubic centimeter times years (f/cc-yrs) or, more simply, fiber years. This accumulated dose determines risk.

   a. Concentration: There has never been competent industrial hygiene evidence that demonstrates working with such products is capable of producing concentrations of etiologic significance during typical use. Thus, evidence of exposure of any potential causal significance to airborne fibers from such materials is lacking.

   b. Time: Time spent working with such products typically is brief (seconds as described in testimony (dep pg 35)), and infrequent. This results in low orders of time; further reducing any potential dose, and risk.

   The combination of insignificant fiber release, if any, and brevity of infrequent occurrence, cannot generate dose of causative disease risk of any asbestos related disease.

2. An inability of the asbestos fiber type possibly used in the Bunsen burner pads or mittens to create mesothelioma risk. Even if Dr. Holinka had continuous exposure to Bunsen burner pads and mittens, there would still have been no risk of mesothelioma.

The term asbestos refers to a group of minerals classified as either serpentine or amphibole on the basis of mineralogical and chemical characteristics. Within the serpentine group there is one type, chrysotile; within the amphibole group there are five: amosite, crocidolite, tremolite, actinolite, and anthophyllite. (Campbell) Just as chrysotile is structurally distinct from the amphiboles (Langer 1989, 1991), these fiber types differ in mesothelioma risk potential.

The scientifically compelling epidemiologic studies and literature reviews that have determined amphibole fiber exposure to be the cause of mesothelioma have also virtually eliminated processed chrysotile as a mesothelioma risk factor. (Acheson, Becklake, Case, Churg (1988, 1994, 2005), Craighead, Hodgson and Damton, Hughes, Lippmann, McDonald (1997, 2002), Rees (1999a,b) Rodelsperger, Roggli (2002, 2004b), Srebro, Thomas, Yarborough)

> "...review of 71 asbestos cohorts exposed to free asbestos fibers does not support the hypothesis that chrysotile, uncontaminated by amphibole substances, causes mesothelioma." (Yarborough)

And as stated in the current edition of a major textbook of pathology:

> "...only amphibole exposure correlates with mesothelioma."
>
> *Husain, AN, Kumar V. The Lung. Ch 15. In: Robbins and Cotran, Pathologic Basis of Disease, 7th Ed., (Kuman, Abbas, Fausto), Elsevier Saunders: Philadelphia, 2007. pg 735.*

It is my understanding that some Bunsen burner pads or mittens may have contained chrysotile asbestos fiber. Processed chrysotile is essentially free of amphiboles. (Frank and Dodson) There is no evidence that amphibole fibers were present in Bunsen burner pads or mittens in this case. The mesothelioma risk associated with amphibole fiber exposure could not have existed with those materials, and could not have been introduced by Bunsen burner pads or mittens.

The factors of inherent lack of contribution to dose and risk, and inability of the asbestos fiber type possibly used in Bunsen burner pads or mittens to create mesothelioma risk eliminates such products from rational consideration as contributors to risk of mesothelioma in this case.

In summary it is my opinion to a high degree of medical and scientific certainty that:

1. Dr. Holinka developed malignant mesothelioma.

2. The mesothelioma in this case should be classified as idiopathic and not asbestos related.

   a. Lack of clinicopathologic evidence.

   b. Lack of history of asbestos exposure considered causal.

   c. The presence of a scientifically plausible alternative etiology; a mesothelioma need not be asbestos related.

   The mesothelioma in this case should be considered idiopathic unless and until appropriate tissue burden studies establish an asbestos etiology.

3. However, if the mesothelioma in this case is erroneously deemed asbestos related, Bunsen burner pads or mittens could not have contributed to risk considering:

a. Inherent lack of contribution to dose and risk

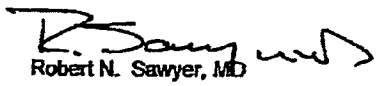
b. An inability of the asbestos fiber type possibly used in Bunsen burner pads or mittens to create mesothelioma risk.

This combination eliminates such products from rational consideration as contributors to mesothelioma risk.

4. Bunsen burner pads or mittens had no role in causation of, or contribution to, the mesothelioma in this case.

Between now and trial, I understand that I may be given an opportunity to review additional material relating to the case. Should any of this additional material alter my opinions; a supplemental report will be provided.

Sincerely,



Robert N. Sawyer, MD

Schedule A

Firm	Defendant
Carol M. Tempesta, Esq MARKS, O'NEILL, O'BRIEN & COURTNEY,P.C. 530 Saw Mill River Road Elmsford, NY 10523	VWR International, Inc. and Univar USA Inc.
Greg Dadika, Esq. REED SMITH LLP Princeton Forrestal Village 136 Main Street, Suite 250 Princeton, New Jersey 0850	ManorCare Health Services, Inc.
Daniel B. Carroll, Esq. DRINKER BIDDLE & REATH LLP 140 Broadway, 38th Floor New York, New York 10005	Baxter Healthcare Corporation
Marc Gaffrey, Esq. HOAGLAND, LONGO, MORAN, DUNST & DOUKAS, LLP 40 Paterson Street New Brunswick, New Jersey 08901 (732) 545-4717	Fisher Scientific International Inc.

References:

Acheson ED, Gardner MJ, Pippard, EC and Grime LP. Mortality of Two Groups of Women Who Manufactured Gas Masks from Chrysotile and Crocidolite Asbestos: A 40-year follow-up. Brit J Ind Med. 1982; 89:344-348.

ACR: American College of Radiology, Asbestos Related Diseases Clinical, Epidemiologic, Pathologic, and Radiologic Characteristic and Manifestations; Chicago, Illinois. 1982.

Battifora H, McCaughey WT. Tumors of the Serosal Membranes. Atlas of Tumor Pathology, 3rd Series Fascicle 15. Washington, D.C., Armed Forces Institute of Pathology, 1995. pg 76.

Becklake MR, Case BW. Fiber Burden and Asbestos-related Lung Disease: Determinants of Dose-Response Relationships. Am J Respir Crit Care Med. 1994; 150:1488-1492.

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Schedule A

Firm	Defendant
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Daniel B. Carroll, Esq. DRINKER BIDDLE & REATH LLP 140 Broadway, 38th Floor New York, New York 10005	Baxter Healthcare Corporation
Marc Gaffrey, Esq. HOAGLAND, LONGO, MORAN, DUNST & DOUKAS, LLP 40 Paterson Street New Brunswick, New Jersey 08901 (732) 545-4717	Fisher Scientific International Inc.

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EDUCATION AND TRAINING

UNDERGRADUATE	Massachusetts Institute of Technology Cambridge, Massachusetts.	BS 1956
MEDICAL	Case Western Reserve University School of Medicine Cleveland, Ohio	MD 1963
INTERNSHIP	Oakland Naval Hospital, Oakland, California. Rotating Internship	1963 - 1964
RESIDENCY	Preventive Medicine. Department of Epidemiology and Public Health. Yale University School of Medicine. New Haven, Connecticut	1967 - 1970
POST DOCTORAL RESEARCH	Yale University School of Medicine, Department of Epidemiology and Public Health. New Haven, Connecticut	1968 - 1970
OTHER DEGREE	Master of Public Health, Yale University School of Medicine, New Haven, Connecticut	MPH 1970
BOARD CERTIFICATIONS	American Board of Preventive Medicine Certificate No. 254, December, 1972	
FELLOWSHIPS	American College of Preventive Medicine. Certificate No. 2023, 1973	
MEDICAL LICENSURE	Connecticut 12054 California G-13270 Colorado 16661	1965 1967 1970
CERTIFICATION	Diplomate of National Board of Medical Examiners. Certificate No. 79307	1964
	Senior Physician, State of Connecticut Certificate No. 312382	1968
	Controlled Substances: Federal Drug Enforcement Administration (DEA) State of Connecticut	 Current Current
SOCIETY MEMBERSHIPS	American Association for the Advancement of Science American College of Occupational and Environmental Medicine	

Occupational and Environmental Medical Association of Connecticut. Offices held: President Elect, Vice President and Member, Board of Directors, National Conference Delegate

New York Academy of Science

FACULTY APPOINTMENTS

Faculty member: Institute for Health and the Environment, Department of Environmental Health Sciences, School of Public Health, University at Albany, State University of New York. 2006

Clinical Professor, Adjunct: Department of Environmental Health and Toxicology, School of Public Health, State University of New York (Albany). 1996 - 2003

Lecturer in Medicine, Extramural Faculty, Oak Ridge Institute for Science and Education, Oak Ridge, TN 1998 - Present

Lecturer in Medicine, Department of Medicine, Associated Faculty, School of Medicine, University of Pennsylvania. Philadelphia, PA 1981 - 1996

Lecture faculty: Mt. Sinai School of Medicine New York, NY 1978-1986

Lecture faculty: The University of Kansas, Division of Continuing Education 1985-1989

Lecture faculty: Department of Epidemiology and Public Health. School of Medicine. Yale University, New Haven, CT 1972-1981

COMMITTEE APPOINTMENTS

U.S. Environmental Protection Agency, Water Engineering Research Laboratory (WERL). Cincinnati, Ohio. Peer Review Committee Chairman. 1987 - 1989

National Institute of Building Sciences. Asbestos Project Committee 1989

National Institute of Building Sciences. Radon Project Committee. 1989

National Institute of Building Sciences. Lead Project Committee. 1989

PRESENT PROFESSIONAL ACTIVITIES

Consultant, Occupational and Preventive Medicine. 1979 - Present

Consultant and Lecturer. Radiation Management Consultants. Philadelphia, PA. Consultation on medical aspects of radiation, care of exposed or contaminated patients.Primary responsibilities: 1979 - Present

Department of Energy Waste Isolation Pilot Project (WIPP), Carlsbad, NM: Emergency response and hospital staff training on plutonium disposal corridors from weapons program sites; radiation effects, treatment.

Federal and state programs lecturer on radio active devices, triage and treatment of injured contaminated patients. Training of hospital staff, emergency responders

Consultant, Entek Environmental and Technical Services, Inc. 1997 - Present
Consultation on medical and biologic aspects of environmental site and structural contamination; including asbestos, lead, molds and fungi, radon, and other potentially toxic agents.

Risk and causation assessment of potential site, structural and personnel exposure.

PAST MAJOR PROFESSIONAL ACTIVITIES

Entek Environmental and Technical Services, Inc., Troy, NY.
Vice President 1986-1996
Chief Executive Officer 1996-1997
Consultants in toxic materials, architectural and engineering services for survey, assessment, and control.

Vice President, Environmental Technology, Inc., 1979-1986
West Hartford, CT. Consultants in toxic material survey, assessment, and control.

Yale University Health Services, Yale University, New Haven, Connecticut. 1972-1981
Head, Preventive and Occupational Medicine.
Surveillance of special risk groups in areas of radiation, carcinogens, biohazards.
Head, Department of Urgent Visit Service. Clinic and in-patient services in primary patient care. Staff included physician, physician associate, and nursing personnel.

Naval Submarine Medical Center, New London, Connecticut. 1969-1972
Staff Medical Officer, Military Operations.
Clinic, hospital, and general medicine duties. Consultation in radiation, chemical toxicology, and infectious disease. Medical Center Command Officer, Senior Medical Officer, Hyperbaric Medicine Officer, and Command Radiation Medical Officer.

Research: Headed section studying relationship of nuclear submarine environment and potential health effects. Directed research in hazardous material control and personnel protection

Post Doctoral Research: Yale University School of Medicine, 1967-1969
School of Epidemiology and Public Health

Principal Investigator in prospective study of Epstein-Barr virus and other respiratory system viral pathogens. Work published in Journal of Infectious Diseases.

Delegate to North Atlantic Treaty Organization (NATO): 1966-1967
Committee on Nuclear, Chemical, and Biologic Effects.

Squadron Medical Officer: U.S. Navy Submarine Squadron Two, 1966-1967
New London, Connecticut.
Clinical out-patient care. Medical system and preventive medicine program development.
Research: Special Projects Medical Officer. Systems development and personnel protection.

	Medical Officer: USS Sam Houston. Polaris Submarine. The Holy Loch, Scotland. General medicine, preventive medicine, and radiation control. Research in epidemiology of infectious diseases in submarine crews.	1964-1966
PAST MAJOR PROFESSIONAL ACTIVITIES (continued)	School of Submarine Medicine: Submarine Medical Center, New London, Connecticut. Medical, surgical, and radiation control training for independent assignment.	1964
	School of Medicine, Western Reserve University. Refer to Training and Education	1959-1963
	Uhl, Hall, and Rich, Architects, Boston, MA Field Office Supervisor, Niagara Hydroelectric Power Project. Niagara Falls, NY	1958-1959
	Union Carbide Corporation; Electrometallurgical Division, Niagara Falls, NY. Engineering Trainee, Special Project Engineer.	1956-1957
	Johnson Controls Corporation, Cambridge, MA. Part time. Drafting and assembly of building and industrial thermal control systems.	1955-1956

PUBLICATIONS

Sawyer, R. Viral serology antibody survival versus storage temperatures available aboard nuclear submarines. Naval Submarine Medical Research Laboratory Report No. 474, 11 March 1966.

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Evans, A.S., Niederman, J.C. and Sawyer, R.N. Prospective studies of a group of Yale University freshman. II. Occurrence of acute respiratory infections and rubella. J Inf Dis 123:3, March 1971.

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PAPERS IN PREPARATION

Sawyer, R.N., Langer, A.M. and Chatfield, E.J. Analysis of a generated complex microparticulate aerosol by analytical transmission electron microscopy.

MAJOR CONSULTATION ACTIVITIES

A list of consultation activities will be provided on written request. The listed consultation activities will be appropriate to the requirements of the request and client confidentiality.

AWARDS

U.S. Navy Surgeon General Award, 1964: Submarine Service (Polaris).

Submarine Force Atlantic Commendation, 1966: Research in toxic materials control and development of medical care systems on nuclear submarines.

U.S. Navy Commendation Medal, 1968: Special projects activities as submarine medical officer.

Bureau of Medicine and Surgery Commendation, 1971: Performance as physician in human hyperbaric research experiments, University of Pennsylvania Institute of Environmental Medicine.

Nominee, Excalibur Award, 1979: Service to the U.S. Government. Environmental Protection Agency.

Nominee, Rockefeller Public Service Award, 1979: Advancing the Health of the American People. Environmental Protection Agency.

PATENT

U.S. Patent No. 5,992,996, 1999: Protective Eyewear Including an Integral Thermoluminescent Dosimetry Chip for Measuring Facial Exposure to Ionizing Radiation.

OTHER ACTIVITY

Fellow, Metropolitan Museum of Art, New York City, N.Y.

CITIZENSHIP United States
BIRTH DATE May 14, 1935
BIRTH PLACE Buffalo, New York

Date of preparation, September 2, 2006 (G)

EXHIBIT L

ENVIRON

June 06, 2007

Ms. Laura Siclari
Hoagland, Longo, Moran, Dunst, & Doukas, LLP
40 Paterson Street, PO Box 480
New Brunswick, NJ 08903

Subject: **Harvey Helfand and Leone Helfand vs. Mannington Mills, Inc.**
Docket Number: 117176-06

Dear Ms. Siclari:

Thank you for referring the above matter to me for epidemiological assessment. It is my understanding that the Plaintiffs claim that Mr. Helfand's pleural mesothelioma resulted from exposure to asbestos from the handling of floor sheeting allegedly obtained from Mannington Mills, Inc.

Following is my expert report in this matter, detailing my credentials, the methods used and materials relied upon, a critical review and synthesis of the relevant epidemiological literature, application of the scientific assessment to the facts apparent in this case, as well as my scientific opinions.

INTRODUCTION

Qualifications

I am by training and experience an epidemiologist. I was trained at the Master's level at the School of Public Health, University of Massachusetts and at the doctoral level at the School of Public Health, University of North Carolina. For ten years I served first as Assistant Professor and then as Associate Professor of Epidemiology in the Department of Biostatistics and Epidemiology of the School of Public Health and Health Sciences, University of Massachusetts. In 1991, I founded Applied Epidemiology, Inc., Amherst, Massachusetts, which in November 2003 merged with ENVIRON International Corporation, where I am a Principal, and serve as Director of Epidemiology. I have special interest and experience in matters pertaining to workplace exposures to various materials and chemicals including asbestos.

I have extensive experience in, for example, designing, conducting and publishing primary epidemiological research; critical review and synthesis of published epidemiological literature; the graduate training of epidemiologists, including classroom teaching, advising and chairing of Master's and Doctoral Committees; and serving in advisory, review and editorial capacities at the local, national and international level. I serve as Adjunct Associate Professor in the Department of Epidemiology, University of North Carolina at Chapel Hill, and Adjunct Associate Professor at Georgetown University, where I have team-taught on several occasions "Epidemiological Applications to Population Health" in the School of Nursing and Health Sciences. I am also an Adjunct Associate Professor and member of the Dean's Advisory Board of the School of Public Health and Health Sciences, University of Massachusetts, where I teach on occasion. I am a Fellow of the American College of Epidemiology, and serve as Vice Chair of the College's Finance Committee. I also serve on the editorial board or as a reviewer of several scientific journals.

I have taught methods for critical review and synthesis of epidemiological studies as part of the core curriculum for Masters and Doctoral candidates in Public Health, and have used these methods to evaluate associations between various exposures and health outcomes. I have applied the same critical review approach to my evaluation of the epidemiological literature on occupational asbestos exposure and risk of mesothelioma, which is summarized below.

A copy of my current Curriculum Vitae which provides additional details as well as a list of my publications is attached.

Overview of approach

I have evaluated the peer-reviewed, published epidemiological literature on the relationship between exposure to various types of asbestos and the risk of mesothelioma, including the consideration of its very long latency (time between exposure and disease occurrence), to determine whether exposure to flooring materials alleged to have been obtained from Mannington Mills was likely to have caused Mr. Helfand's pleural mesothelioma. I have employed standard and widely accepted methods for critically and comprehensively reviewing and synthesizing the published, peer-reviewed epidemiological literature, and formulated my scientific opinions and conclusions based on this analysis. In addition to the relevant peer-reviewed, published epidemiological literature, I draw upon my education, training and professional experience, as well as on my analysis of materials provided to me by counsel for Defense, including but not limited to the complaint, interrogatory responses and deposition transcripts, to formulate my professional opinions and the conclusions offered. A bibliography of all materials relied upon is included below. I expect to review and comment upon additional scientific publications, documents, testimony, expert reports, exhibits and discovery related to the topics of this report as they become available.

METHODS

The epidemiological approach

Epidemiology is the field of public health that includes the study of incidence, prevalence, and distribution of disease in human populations, and factors that may be related to disease occurrence. It is a science that employs standard methods to identify and interpret statistical correlations, called "associations," between disease occurrence and other factors. Epidemiological research results are central to the determination of the role of specific risk factors in the general causation of disease in humans, and are broadly relied upon by epidemiologists and other professions as a basis for decision-making, including development of policy and judgments regarding specific causation. The validity of the epidemiological evidence and the validity of its interpretation determine the reasonableness of causal judgments that rely upon such evidence.

Epidemiological research addresses whether a disease is associated with specific exposures in groups of people or populations. Exposures measured on an individual basis provide the strongest evidence of an association, if it exists, between the exposure of interest and disease. Analytic techniques also can control for, or eliminate possible effects of other exposures that may be related to the exposure of interest, or the disease under investigation.

Epidemiologists are generally concerned with the impact that bias, due to systematic error, might have on study results. Systematic error in the methodology or due to missing or inaccurate information can render results invalid or even misleading, possibly related to how the groups being compared have been defined. Factors determining the quality of epidemiological studies include the ability to avoid biases such as selective participation of certain subsets of individuals (selection bias), systematic errors in responses or measured data (information or misclassification bias), and identification of other strong risk factors for the same outcome that are correlated with the factor of interest (confounding bias). Though the potential for bias exists in all studies, some study approaches and research settings are more prone to bias than others. The degree to which these challenges are addressed and overcome in an epidemiological study partly determines the strength and validity of the study results.

Interpretation of the epidemiological literature also considers the role of chance in the results. Statisticians and epidemiologists evaluate the probability that an observed result is due to chance by applying tests of statistical significance. If the results are not statistically significant, chance cannot reasonably be ruled out as an explanation for the reported association (i.e., accepting a 5% error rate).

Dose-response assessment, or the evaluation of the relationship between estimated or actual dose of exposure and the disease risk, is a key tool of epidemiology. Ideally, the dose estimate is derived based on a reasonable period of disease latency. Latency is usually described as the time elapsed between the first known exposure to the agent of interest (often indicated by date first employed in a particular job where exposure is likely) and the diagnosis of the disease of interest. This is also known as maximum latency, and is influenced by the ability to validly identify first exposure as well as the

ability to detect the disease soon after it occurs. For many cancers that produce solid tumors, the latency is usually 20 or more years, and can exceed 50 years (as with mesotheliomas). If exposure is assessed without regard to disease latency, some or all of the exposure evaluation may be irrelevant to the occurrence of the disease.

Assessments of dose-response and latency are frequently used to inform causal judgment and policy formulation. The greatest impediment to such analyses is that many epidemiological studies fail to, or cannot, accurately characterize first possible exposure and the specific level or concentration of exposure for each individual in the study: in fact, most studies that consider exposure rely upon surrogate measures such as employment history.

In addition to assessing the methodological quality of individual studies, weight of evidence syntheses consider the overall breadth and quality of the literature available. Just as individual studies might be subject to systematic biases, a body of literature might be biased because of its focus on a chosen or preferred research hypothesis or study approach, or as a result of selection for publication. Such "publication bias" occurs when authors preferentially submit, and journals preferentially accept, studies demonstrating positive findings, even if such studies may be positive due to methodological weakness (e.g., small study size) or errors (e.g., exposure misclassification). Null study findings, even if based on well-designed and conducted studies, are considered "less interesting" and are less likely to be published (Hennekens and Buring 1987). The overall bias will be exaggerated if null findings contradict positive findings and/or if a weak study supports (or replicates) previously "accepted" associations.

Use of epidemiology for judgments of disease causation

Where a balanced and complete literature is available, it is possible to characterize that literature as reasonably establishing an association that is or is not probably causal. If the weight of evidence favors a judgment of general causation, i.e., that a risk factor under certain conditions is capable of causing the disease in adequately exposed persons, epidemiological research can further help determine whether specific exposure attributes and other risk factors are more or less likely to contribute to risk among exposed persons. These risk factors may include but are not limited to the following:

- Dose or exposure concentration
- Type or form of exposure (chemical composition or physical structure)
- Timing of exposure (year first exposed, duration of exposure, etc.)
- Host susceptibility (genetic polymorphisms)
- Host attributes (age, sex, ethnicity, etc.)
- Co-exposures (viruses, smoking, etc.)
- Co-morbidity

Epidemiology generally cannot directly ascertain specific causation, i.e., whether a specific exposure played any role in the disease of specific individuals. Some cases of disease are idiopathic, and arise regardless of an individual's exposure history. However, understanding at a group level those factors that are associated with increased risk of disease may improve our ability to identify high or higher risk groups so that reductions

in exposure can be targeted and implemented to prevent future disease. Based on these methods, we may also improve our ability to determine which subgroups are more likely to be at risk due to their exposures, and by extension (and for non-scientific purposes such as decision-making and litigation) determine that the relationships observed at the group level may reasonably be applied to individuals who are in the study group or an adequately similar population to which such results may be applied. The determination of causation, however, remains a judgment, and cannot be proven. Although direct experimental evidence may offer stronger support for causal inference, experiments on health effects of toxic materials on humans are unethical. Toxicological or other laboratory data collected from animal models are useful for understanding the mechanisms of effects and for developing hypotheses regarding human health effects, but are not directly applicable to humans. For these reasons, well-designed epidemiological studies have been identified as the preferred scientific support for regulatory agencies such as the Environmental Protection Agency (EPA) and the Occupational Safety and Health Administration (OSHA), which determines policies regarding possible adverse effects of exposures. For the same reasons, the determination that an association is causal in humans is most reasonably based on the availability of and proper evaluation of good epidemiological data.

Epidemiological review of asbestos and malignant mesothelioma

For this report, PubMed was used to identify key research reports and reviews published in the peer-reviewed medical/health literature that are relevant to the topics of this case. Provided by the US National Library of Medicine, PubMed is a powerful standard research tool available free over the internet, which searches the medical literature published since about 1966. The keywords "asbestos," "crocidolite," "amosite," "amphibole," "chrysotile," "pleural," "mesothelioma," "occupation," "dust," "printing," "lithographic," "epidemiology," and "exposure assessment" were entered in various forms and combinations to identify a universe of potentially relevant articles available in the English language. Abstracts of the articles identified were screened for relevance, and the full article for those meeting at least minimal criteria (epidemiological study, standard methods utilized, study population asbestos-exposed, analyses conducted by occupational risk factors such as asbestos exposure level, duration of exposure, type of industry, type of asbestos present, or other risk factors that help explain the risk of mesothelioma) were more thoroughly evaluated. Occupational studies of the primary and secondary asbestos industries were included and are summarized below, as these studies examine populations of workers with documented and often heavy asbestos exposures and provide a considerable basis for understanding and evaluating potential risks among groups with lower exposures, including presumed and background level exposures.

EPIDEMIOLOGY OF ASBESTOS AND MALIGNANT MESOTHELIOMAS

Overview

Malignant mesotheliomas are cancers that arise most often in the pleura, the thin layer of tissue that surrounds the lungs and lines the chest cavity, or the peritoneum, the thin layer of tissue that surrounds the abdominal cavity and lines the abdominal organs (Blot and

Fraumeni 1996). The risk of all types of mesothelioma combined is about 11 per million population per year in the US, with about 3,000 new cases diagnosed each year (Antman, Hassan et al. 2005). The incidence of cases not thought to be due to occupational exposure has been estimated at 1-2 cases per million person years (Bertazzi 2005).

Tumor registry data show an increase in the number of all types of malignant mesotheliomas, combined, beginning in the 1950s (Blot and Fraumeni 1996). Incidence increased sharply beginning in the 1970s and continued to rise through the mid-1990s (Price and Ware 2004; Bertazzi 2005). The increase has occurred almost exclusively among white men; the rate for women in the U.S. has been stable since the 1970s at about 5 per million per year. Rates for non-white men have also remained relatively stable over time (Bertazzi 2005).

Risk factors for mesotheliomas include exposure to amphibole asbestos, erionite (a fibrous mineral from the zeolite group) and thorium dioxide (Thorotrast) – a radioactive contrast medium used in X-ray diagnostics until the 1950s in the U.S. "Amphibole" refers to a group of five of the six fibrous minerals that are considered to be asbestos: crocidolite, amosite, anthophyllite, actinolite, and tremolite. Amphiboles are characterized by thin, rod-like fibers. In contrast, the most widely used form of asbestos, chrysotile, is a serpentine mineral, with curly and pliable fibers. Simian Virus 40 (SV40) cells have been discovered in mesothelioma tumors, suggesting the virus might play some role in the development of mesothelioma (Carbone, Rizzo et al. 2000; Vilchez, Kozinetz et al. 2003). Neither smoking nor ionizing radiation is considered a risk factor for mesothelioma (Antman, Hassan et al. 2005).

Some mesotheliomas are idiopathic, meaning they are not due to exposure to any of the recognized risk factors for the disease. Estimates of the rate of idiopathic mesothelioma may be derived from population-based cohort studies, where between 10% and 50% of mesotheliomas detected occurred in the absence of identifiable asbestos exposure (Boffetta 1998; Antman, Hassan et al. 2005; Bertazzi 2005). A proportion of all occupationally asbestos-exposed individuals with mesothelioma are also believed to develop mesothelioma due to causes other than their occupational asbestos exposure. Some number of idiopathic cases is expected to occur even among individuals with a history of substantial amphibole asbestos exposure.

Asbestos exposure and risk of pleural mesothelioma

Among asbestos-exposed persons, the chief factors that influence the risk of pleural mesothelioma are asbestos fiber type, the time since first exposure to asbestos (latency), and exposure (dose).

Exposure to amosite, crocidolite or other types of amphibole asbestos is the clearest risk factor for pleural mesothelioma. In contrast, epidemiological evidence suggests the association between chrysotile asbestos exposure and pleural mesothelioma risk, if any, is weak.

The epidemiological literature indicates that average latency – i.e., the time between first adequately high exposure to amphibole asbestos and occurrence of mesotheliomas – is

very long, and may be as much as 60 years. The duration of latency may depend on both dose and fiber type. Low exposures are generally associated with longer latencies. Occupational studies of amphibole exposure and mesotheliomas typically show a dose-response relationship, with low or no risks observed among those with the lowest exposures.

Low-level asbestos exposures are also ubiquitous in the general population (i.e., not occupationally exposed persons), and can arise from regional geological features and from fibers released into the environment from consumer and building products. Use of asbestos-containing (predominantly chrysotile-containing) consumer and building products increased after World War II through the 1970s, at which time a ban went into effect on asbestos in some consumer products, and asbestos was voluntarily withdrawn from other products. Ambient asbestos exposures also are thought to have increased and then declined over this interval, parallel to the increase and decrease in use of asbestos-containing consumer goods, though ambient environmental levels remained far below those measured in some work places. The lack of an increase over time in mesothelioma risk among women and non-white men has been taken as evidence for a necessary threshold, greater than ambient environmental levels, for amphibole asbestos exposure to lead to increased risk of mesothelioma (Bertazzi 2005).

Occupational studies of primary asbestos exposure and mesothelioma

All primary asbestos industries have reported increased mortality from pleural mesothelioma or all types of mesothelioma, combined. The primary asbestos industries include **mining and milling** (de Klerk, Armstrong et al. 1989; Piolatto, Negri et al. 1990; Sluis-Cremer, Liddell et al. 1992; Liddell, McDonald et al. 1997), **asbestos cement manufacturing** (Thomas, Benjamin et al. 1982; Finkelstein 1984; Alies-Patin and Valleron 1985; Ohlson and Hogstedt 1985; Gardner, Winter et al. 1986; Hughes, Weill et al. 1987; Raffn, Lynge et al. 1989; Albin, Jakobsson et al. 1990; Neuberger and Kundi 1990), **textile manufacturing** (McDonald, Fry et al. 1983; McDonald, Fry et al. 1983; Peto, Doll et al. 1985; Dement, Brown et al. 1994), **insulating** (Seidman, Selikoff et al. 1986; Seidman and Selikoff 1990), **friction and insulation materials manufacturing** (McDonald, Fry et al. 1984; Enterline, Hartley et al. 1987; Newhouse and Sullivan 1989); and **filter assembly and manufacturing** (McDonald, Gibbs et al. 1978; Jones, Smith et al. 1980; Talcott, Thurber et al. 1989) . Studies conducted in the primary asbestos industries, where the broadest range of exposures can be found, offer the best opportunity to quantify mesothelioma risks.

Mesothelioma risk depends on asbestos fiber type

Chrysotile accounts for 95% of asbestos produced in the world (Harington 1991; Terracini 2006) and it is the most commonly used asbestos fiber in the US. As noted above, however, risk of mesothelioma is much more strongly associated with amphibole asbestos exposure. Among the amphiboles, crocidolite is most strongly associated with mesothelioma risk. Intermediate risks are seen among workers exposed to amosite. Increased risk of mesothelioma from occupational exposure to chrysotile alone is less apparent and not seen in many studies. The risk differential for mesothelioma at similar exposure levels of chrysotile: amosite: crocidolite has been estimated to be 1:100:500

(McDonald and McDonald 1996; Hodgson and Darnton 2000). In studies where mesothelioma risk appears to be increased among workers with high-level chrysotile exposures (e.g., asbestos miners), there is reasonable evidence that the risk derives from contamination by amphibole fibers, as comparable groups heavily exposed to pure chrysotile appear not to be at increased risk of mesothelioma (McDonald and McDonald, 1996).

Crocidolite asbestos

In employees with predominantly or heavy crocidolite exposure, the relative risk of pleural mesothelioma compared to unexposed or less exposed employees is extremely high, even while the absolute risk remains low. For example, proportional mortality from mesothelioma was examined among 33 men employed during 1953 in a Massachusetts factory that manufactured cigarette filters containing crocidolite. Through 1988, a total of 28 deaths had occurred. Five were from pleural mesothelioma, with only 0.01 expected (RR=460; 95% CI 150, 1080) (Talcott, Thurber et al. 1989). Jones et al. investigated causes of death among 951 women who had used crocidolite to manufacture military and commercial grade gas mask filters during the 1940s. The authors reported 29/166 deaths were due to mesothelioma, with risk positively associated with duration of exposure. The most deaths in this group occurred during the middle 1970s, approximately 30 years after first exposure to crocidolite (Jones, Smith et al. 1980).

Initial reports for a cohort of 13,450 friction product workers who primarily manufactured brakes and brake material between 1941 and 1979 identified 10 deaths from mesothelioma, 9 among those with known crocidolite exposure. There was no excess among those exposed to chrysotile, the predominant fiber type used at the factory (Newhouse, Berry et al. 1982; Berry and Newhouse 1983). An updated mortality study of the cohort reported a total of 13 mesothelioma deaths, 11 among those known to be exposed to crocidolite (Newhouse and Sullivan 1989).

Chrysotile asbestos

Studies of workers predominantly exposed to chrysotile fibers inconsistently demonstrated slightly elevated risks of mesothelioma mortality compared to non-exposed populations. McDonald et al. identified 4,137 male textile workers who were mainly exposed to chrysotile who were employed for more than one month between 1938 and 1958 at a plant in Pennsylvania. Fourteen out of 1,392 death certificates mentioned mesothelioma (McDonald, Fry et al. 1983). Dement et al. (1994) reported mortality for a similar South Carolina textile manufacturing plant cohort, including those employed for at least one month between 1940 and 1965, with vital status ascertained through 1990 (Dement, Brown et al. 1994). Two pleural mesothelioma deaths were reported among workers primarily employed in the spinning department, with latency of 37 and 34 years, and 25 and 32 years employment, respectively. A third pleural mesothelioma death was identified when mortality follow-up was extended through 2001. The most recent decedent also had been employed in the spinning department, with a latency interval of nearly 50 years (Hein, Stayner et al. 2007).

Among 1,058 Italian chrysotile asbestos miners employed since 1946, two deaths due to pleural mesothelioma were identified among 427 total deaths – one in a worker with 20 to 30 years since first exposure and the other in a worker with more than 30 years since

first exposure (Piolatto, Negri et al. 1990). One pleural mesothelioma was reported in a study of 2,167 chrysotile asbestos cement workers (Gardner, Winter et al. 1986) and no mesothelioma deaths were reported by McDonald et al. (1984) among 3,641 US friction product workers where chrysotile was the predominant fiber used (McDonald, Fry et al. 1984).

It remains unclear whether the occasional mesothelioma cases seen among heavily chrysotile-exposed workers are due to high-intensity chrysotile fiber exposure, amphibole fiber contaminants, unrecognized amphibole exposure from other sources, or are of idiopathic origin.

Mixed fiber types

Workers handling primarily one type of asbestos may unwittingly have exposure to other types, or may come to a specific workplace with prior exposure to amphibole asbestos. Also, as mentioned above, chrysotile may be contaminated with amphibole fibers. When mesothelioma risk among primarily chrysotile-exposed occupational cohorts has been identified, it has generally not been possible to determine if the risk was due to chrysotile, contamination with amphiboles, other sources of amphibole exposure, or from some combination of these (Elmes 1994; Britton 2002). For example, the Pennsylvania textile workers described above were mainly exposed to chrysotile, but crocidolite and amosite were also present in the plant. Although information about exposure to specific fiber types was not provided in the report, the authors attributed the mesothelioma cases to amphibole exposure (McDonald et al., 1983b).

Prior to World War II, insulation workers in New York and New Jersey were primarily exposed to chrysotile fibers. Subsequently, amosite was added to some of the insulation. In a cohort study of New York and New Jersey insulators, those working for long periods after the war were most likely exposed to amosite, an amphibole, and were at substantially increased risk of mesothelioma. No increase in risk was observed among those only exposed to chrysotile prior to the war, or those exposed to both chrysotile and amosite after the war but for less than 20 years (Selikoff, Hammond et al. 1979). This latter observation suggests that some exposure threshold (concentration and duration) may be required before risk is meaningfully elevated; however, evidence of such a threshold has not been consistently observed.

Yarborough also reviewed studies of occupational cohorts with exposures to mixtures of fiber types (Yarborough 2006). The cohorts, including some from the studies cited above, had a total of 32,000 employees with exposures thought to be relatively pure chrysotile. Among these, only seven mesothelioma cases were identified, and Yarborough found reason to question the accuracy and adequacy of the exposure evaluation and/or diagnosis for each of them.

Lung fiber burden analyses generally indicate exposures to mixtures of fiber types, even if work histories are unable to document mixed exposures (Yarborough 2006). For example, McDonald et al., (1997) examined dried lung specimens from chrysotile miners and millers in Quebec who died of mesothelioma. Of the 27 mesothelioma cases, 21 dried lung specimens were available. The investigators found tremolite and chrysotile fibers in 14 specimens from cases who worked in the Thetford mines, and chrysotile,

tremolite, crocidolite and amosite in specimens obtained from the remaining 7 cases who worked in the mines in Asbestos (McDonald, Case et al. 1997). This investigation indicates that mesotheliomas that were attributed to chrysotile exposure may be due to the amphiboles contaminating the chrysotile at specific mining sites.

Although they offer suggestive evidence, lung fiber burden studies can be criticized on the grounds that fibers found in the lung at autopsy have an unknown relationship to fiber exposures that actually led to mesothelioma induction. Epidemiological evidence suggests that at least 30 years, and perhaps as much as 50-60 years, must elapse between initial exposure and the onset of mesothelioma (see discussion of latency, below). Furthermore, amphiboles are expected to be found many years after exposure has ceased due to their persistence in lung tissue, whereas chrysotile fibers are relatively rapidly broken down and eliminated (Churg and DePaoli 1988; Britton 2002; Bernstein and Hoskins 2006). It is the relative durability and persistence of the amphiboles that is believed to contribute to their carcinogenic potency.

Exposure (Dose)

The relationship between mesothelioma and either intensity or duration of exposure, or a cumulative (intensity and duration combined) estimate, is complex. Because exposure measurements are frequently unavailable in epidemiological studies, duration of employment or other surrogates are often used as indicators of "dose." Occupational studies that adequately account for disease latency and fiber type generally report a positive association between mesothelioma risk and either duration of employment or quantitative dose estimates. For example, of 8,009 deaths investigated among workers in the asbestos mines in Quebec, 22 of 25 mesothelioma cases were among men employed 20 or more years in the Thetford mines, and 5 additional cases were among men employed for at least 30 years in the Asbestos mines (McDonald et al., 1997). In a later publication, it was reported that the rate of mesothelioma increased with increasing fiber-years of exposure (Liddell, McDonald et al. 1997). Among 6,506 crocidolite miners/millers in Western Australia, the relative risk was 10.5 (95% CI 3.12-35.1) for pleural mesothelioma among those with more than 6 months of exposure compared to those with shorter duration of exposure (de Klerk, Armstrong et al. 1989).

Among asbestos cement workers, Albin et al (1990) reported an increasing risk of pleural mesothelioma with increasing cumulative exposure compared to unexposed workers: RR=1.9 for workers with an average dose of 3.1 f/ml-years; RR=21.2 for an average dose of 25.6 f/ml-years; and RR=22.8 for average dose of 88.2 f/ml-years (Albin, Jakobsson et al. 1990). Similarly, Finkelstein (1984) reported an increasing trend in mesothelioma (all types) mortality with increasing dose among asbestos cement workers. The mean cumulative exposure for eleven pleural mesothelioma cases was 42 f/ml-years, and 161 f/ml-years for eight peritoneal mesothelioma cases (Finkelstein 1984). Among amosite-exposed factory workers, the lower the dose (time worked), the longer the time required for development of disease (Seidman, Selikoff et al. 1986).

Fiber type remains an important consideration when assessing the risk of mesothelioma associated with exposure concentration or duration. Among a cohort of South African miners, workers were exposed to crocidolite, amosite, or mixtures of both fibers (Sluis-

Cremer, Liddell et al. 1992). Of the 30 cases of mesothelioma identified, 20 occurred among those with crocidolite exposure (none with less than 12 months exposure), 4 among those exposed to amosite for more than 3 months, and 6 among those with mixed exposures for more than 3 months. Cumulative exposure averaged 15.2 fibers/ml/year for the amosite group compared to 9.6 fibers/ml/year for the crocidolite group.

Time since initial exposure (Latency)

Time since initial exposure to amphibole asbestos is a strong predictor of incidence of mesothelioma. In the PMR study of cigarette filter manufacturers described above, the median interval from first exposure to crocidolite asbestos until mesothelioma death was 34 years (range, 26 to 37) (Talcott, Thurber et al. 1989). Any mesothelioma deaths that occurred after 1988 would only increase the average estimated latency, as cases with longer latency would not yet have been detected as of 1988, when the study was completed. In general, when mortality is used instead of disease incidence or diagnosis, the overall duration of latency will be somewhat exaggerated. Because the average survival time after mesothelioma diagnosis is short (about 1 year) (Stewart, Edwards et al. 2004; Antman, Hassan et al. 2005), the effect of using death rather than incidence or diagnosis to calculate estimated latency for mesothelioma will be minimal

In Australia, the incidence of malignant mesothelioma lagged 20 to 30 years behind trends of amphibole (primarily crocidolite) exposure. Incidence of mesothelioma for 1964 to 1968 among those 35 years or older at diagnosis was less than 1.0 case per million person-years, increasing to 15.5 cases per million person-years in 1979-1980. Among 65- to 74-year-old men in 1979-1980, mesothelioma occurred in 69.7 cases per million person-years (Musk, Dolin et al. 1989).

Among 10,918 chrysotile miners and millers in Quebec first employed in 1904 and followed until 1992, a total of 38 mesotheliomas were reported – 33 in miners and millers and 5 in factory workers. Of these, 21 occurred in workers from the Thetford mines and in Asbestos mines and mills – both sites among the earliest operations in Quebec (Liddell 1997). Average latency was 47 years, with a range of 21 to 60 years. PMRs increased with year of death: no mesothelioma deaths occurred before 1950, but subsequent mesothelioma death rates were 0.18% (1950-1974), 0.68% (1975-1984) and 1.10% (1985-1992). Rates of mesothelioma were significantly greater at the oldest Thetford mines (35.3/100,000 subject years) than at the Asbestos mine and mills (13.2/100,000) (McDonald, Case et al. 1997), possibly because of the longer time follow-up time.

A mortality study of 4,137 textile manufacturing workers employed more than one month between 1938 and 1974 and exposed to chrysotile, amosite and crocidolite identified 14 mesothelioma deaths between 1960 and 1975. With the exception of one subject, deaths occurred 25 to 53 years from first employment (McDonald, Fry et al. 1983).

Initial studies of the insulation workers in New York and New Jersey followed through 1976 reported no mesotheliomas among those with less than 20 years exposure; 7 mesotheliomas occurred among those with 20 to 34 years, and 31 mesothelioma cases in New York/New Jersey insulators occurred after 35 years of exposure. Among a larger cohort of US and Canadian insulation workers followed through the same period, only 5

of 224 mesotheliomas occurred between 5 and 19 years after onset of exposure; rates of pleural mesothelioma were 2.78 per1,000 person-years for 35 to 39 years after onset and 5.47 per1,000 person-years for peritoneal mesothelioma 45 or more years after exposure onset. Additional follow-up of the 17,800 asbestos insulation worker cohort found peak mortality from mesothelioma (5.1 per 1,000) occurred 45 years after first employment. Although excess deaths from all causes combined decreased over time, there was no apparent decrease in deaths due to pleural mesothelioma or for those with more than 40 years since onset of first exposure (Seidman and Selikoff 1990).

Based on these studies, it is apparent that the greatest risk of mesothelioma occurs on average 40-50 years after first substantial exposure to amphibole asbestos occurs. Lower exposure concentrations would be expected to require even longer latency.

Studies of secondary asbestos exposure and mesothelioma

Construction trades

Mesothelioma risk has been described among employees in various construction trades due to their use of asbestos-containing insulation and building materials. Historically, exposure to asbestos in the building / construction industry occurred during installation and removal of insulation, installation of duct work for ventilation, among plumbers and electricians cutting into or disrupting installed asbestos insulation, and during plaster preparation/mixing and dry sanding. More recently, chrysotile asbestos exposures in construction may occur – but at much lower levels and almost entirely limited to chrysotile fibers – in the process of handling and installing drywall joint compound, roofing and siding materials, as well as some flooring materials. Higher exposures, and therefore greater mesothelioma risks, are likely to result from the removal, demolition or rehabilitation of older construction containing asbestos insulation or other friable amphibole-containing materials than in the installation of new, non-friable chrysotile-containing materials such as roofing and flooring materials, etc. Many building/construction products contained small amounts of chrysotile asbestos until around 1975, when most manufacturers reduced and eliminated asbestos in response to a ban on asbestos in some such products. The specific asbestos fiber type that may have been used in a particular product is often unknown (Huncharek 1992). More recently, the likelihood of asbestos exposure due to the use and handling of new building materials has been greatly reduced.

Several studies have reported risk of mesothelioma among construction workers (Robinson 1995; Wang, Dement et al. 1999; Stern, Lehman et al. 2001; Koskinen, Pukkala et al. 2002; Engholm and Englund 2005). For example, Robinson and colleagues (1995) studied the mortality experience of 61,682 men who died between 1984 and 1986 in 19 states, employed in various occupations in the construction industry, using usual occupation as reported on the death certificate. Cancers of the pleura and peritoneum were observed for carpenters (PMR=163; 95% CI 89, 274, based on 14 deaths), plumbers (PMR=327; 95% CI 106, 763, based on 5 deaths), insulation workers (PMR=2467 based on 2 deaths), and electricians (PMR=331; 95% CI 122, 719 based on 6 deaths). The largest subgroup of the cohort was construction laborers, with nearly 10,000 men included; although other causes of death were elevated in this subgroup, there were no mesothelioma deaths noted (Robinson 1995).

Koskinen et al. (2002) followed 16,696 Finnish construction workers participating in cancer screening campaign from 1990 through 2000. Of 1,320 incident cancers, 13 mesotheliomas generated roughly a doubling of risk (SIR=1.96; 95% CI 1.04, 3.35). The only occupational sub-groups with significantly elevated risks included insulators and electricians, with 3 and 4 mesotheliomas in each group, respectively (Koskinen, Pukkala et al. 2002).

Flooring mechanics

Epidemiological studies of construction industry employees include floor installers and removers. When construction occupations have been examined separately by trade, floor installers and removers have not been identified among those at increased risk of mesothelioma (Robinson 1995; Wang, Dement et al. 1999; Koskinen, Pukkala et al. 2002; Engholm and Englund 2005). Specific studies of mesothelioma or other asbestos-related risks focusing exclusively on floor installers and removers have not been identified.

Some estimates of exposure during standard floor removal procedures are available, and indicate very low fiber concentrations. Lange et al (1996) reported that exposures due to floor tile removal resulted in arithmetic and geometric mean airborne fiber concentrations 0.005 f/cm^3 (range: 0.005 to 0.01 f/cm^3) (Lange, Lange et al. 1996). Similarly, Williams and Crossman reported fiber concentrations resulting from breaking vinyl asbestos floor tiles with a hammer were at or below detection limits for OSHA-specified monitoring methods (i.e., exposures were <0.0058 structures/cm^3) (Williams and Crossman 2003).

Printing

Several potentially hazardous exposures are known to occur in the printing industry, including heavy metals, solvents, mineral oils, carbon black and asbestos (Partanen, Heikkilä et al. 1991; IARC 1996; Tolbert 1997; Bulbulyan, Ilychova et al. 1999), and increased risks of some cancers have been reported (Partanen, Heikkilä et al. 1991; Morabia, Markowitz et al. 1992; Lynge, Rix et al. 1995; Tolbert 1997; Cocco, Dosemeci et al. 1998; Cocco, Ward et al. 1998; Bulbulyan, Ilychova et al. 1999; Jahn, Ahrens et al. 1999; Rafnsson 2001). Following a 1996 review, the International Agency for Research on Cancer reported that "occupational exposures in printing processes are possibly carcinogenic to humans" (IARC 1996). Mesothelioma, however, was not among the outcomes identified.

Summary of occupational studies of secondary asbestos exposure in construction trades

Construction workers may be exposed to low levels of asbestos intermittently over a long period of time (the duration of their employment), and have been observed to be at low risk of mesothelioma. The specific exposure levels depend on trade, and occupations within the construction industry can be roughly ranked according to potential exposure. Plumbers and electricians tend to be most highly exposed; those involved with installation and removal of asbestos containing roofing and siding products are generally at lower risk, followed by wallboard installers and plasterers. Flooring mechanics typically do not generate high levels of asbestos exposure and have not been reported in the occupational health literature to have elevated mesothelioma risk. The vast majority of asbestos used in construction materials is chrysotile, which has not been convincingly

associated with risk for mesothelioma. The duration of the latency interval appears to be proportional to the intensity of amphibole exposure, with lower-intensity exposures requiring latency intervals of as much as 50 years or longer.

Although amphibole asbestos exposure is the most likely risk factor for mesothelioma, information on prior asbestos exposures of any kind may be sought specifically when mesothelioma is diagnosed. Information on specific occupational and other sources of asbestos exposure may be less actively sought for diseases or causes of death that have not been strongly associated with a specific exposure. Actively searching for any source of asbestos exposure when a diagnosis of mesothelioma is generated, or differentially seeking such information from cases and controls, would erroneously create or exaggerate observed associations between mesothelioma and asbestos exposure. These forms of bias may most impact studies of occupational groups less clearly or consistently exposed to amphibole asbestos.

SUMMARY OF RELEVANT FACTORS IN THIS CASE

Mr. Helfand was diagnosed with malignant pleural mesothelioma in October 2006. Based on an extensive and critical evaluation of the epidemiological literature, the main determinants of risk of pleural mesothelioma include exposure to amphibole asbestos fibers, with risk dependent both on exposure intensity and latency, with "latency" defined as the time between first exposure to substantial amphibole asbestos and the diagnosis of disease. Intermediate risks are noted among those exposed to mixed fiber, presumably in proportion to the concentration of amphibole asbestos fiber present. However, the literature also indicates that mesotheliomas occur in substantial proportion among persons with no significant exposure to amphibole asbestos fibers. These cases may be idiopathic or the result of unrecognized exposures to amphibole asbestos sources that might have occurred 40-60 years prior to diagnosis. Understanding these factors – the intensity, duration and timing of exposure to specific asbestos fiber types – as they pertain to Mr. Helfand's exposure history and disease occurrence is important in evaluating whether specific potential sources can reasonably be considered causes of his mesothelioma.

According to his deposition testimony, Mr. Helfand completed approximately 50-100 home improvement projects from 1951 until approximately 2005. About 25-30% of these jobs involved flooring work, but of these the majority (i.e., 90%) of the flooring projects he completed involved tile. Mr. Helfand reported only installing Manning Mills flooring and did not recall removing any Mannington Mills products.

Mr. Helfand alleged that since the late 1950's he installed non-tile flooring products including 'linoleum' and 'sheeting' goods manufactured by Mannington Mills, and testified that he used a total of approximately five rolls of Mannington Mills sheeting. Mr. Helfand testified that he was exposed to asbestos dust and fiber when he cut the sheet material to dimensions needed for each installation project. Cutting flooring potentially containing asbestos (typically in the backing) would have been of short duration, would have occurred rarely, and would have generated negligible to very low levels of respirable fiber. Mannington Mills produced their first sheet flooring in late 1962, and

introduced chrysotile in the backing of some products in 1963. While some products contained chrysotile asbestos in the backing material, the asbestos was encased in a latex binder, and therefore would release minimal fibers when cut. Asbestos was removed from Mannington Mills products in the early 1980s. Thus, the only asbestos fiber type expected to be released from cutting sheet goods would have been chrysotile. Brief, infrequent exposure to low concentrations of chrysotile asbestos fibers would not have contributed to an increased mesothelioma risk.

Mr. Helfand also testified that he was exposed to asbestos while personally handling Linotype machines around 1950. Working as a clean-up boy after school and during the summer, Mr. Helfand described exposure to asbestos paste while replacing the linings of pots used for melting lead. Mr. Helfand continued to work in the printing industry after graduating from NY School of Printing in 1953 at various jobs and locations throughout his life, until 2005. According to his testimony, Mr. Helfand performed hands-on work at various printing-related jobs including: helper, apprentice, pressman, and foreman. These jobs entailed duties such as cleaning, operating and maintaining presses, quality control, production and general supervision. Mr. Helfand alleged that he was exposed to asbestos while performing some of these duties. It is not known, however, whether amphibole asbestos was present or if appreciable exposures resulted from these activities.

CONCLUSIONS

Based on my review, analysis and synthesis of the published epidemiological, occupational health and case-specific information available to me, I conclude to a reasonable degree of epidemiological certainty that Mr. Helfand's limited handling and cutting of floor sheeting goods produced by Mannington Mills – some of which potentially contained chrysotile asbestos – did not cause Mr. Helfand's mesothelioma. The greatest and most consistently increased risks of pleural mesothelioma reported in the published epidemiological literature are associated with substantial amphibole asbestos exposures. Nevertheless the literature also indicates that substantial numbers of idiopathic pleural mesotheliomas do occur, and would be expected regardless of asbestos exposure. However, whether his disease was truly idiopathic or a result of some undocumented exposure to amphibole asbestos exposure cannot be ascertained. Mr. Helfand's limited and sporadic potential exposure to low-levels of chrysotile asbestos generated by cutting Mannington Mills sheeting goods would have been negligible, if present, and of no consequence with respect to his pleural mesothelioma.

Please do not hesitate to contact me if you have questions or require further information.

Sincerely yours,

Kenneth A. Mundt, Ph.D.
Principal and Director of Epidemiology
ENVIRON International Corporation

Case-specific materials relied upon

Document title	Description
Complaint	Complaint
Helfand Depo Vol 1	Deposition of Harvey Helfand, Volume 1
Helfand Depo Vol 2	Deposition of Harvey Helfand, Volume 2
Helfand Depo Vol 3	Deposition of Harvey Helfand, Volume 3
Plaintiffs Answers to Interrog	Plaintiff's Answers to Interrogatories
SSN Records Helfand Medical Reports	Social Security Records Expert Report of E. Neil Schachter, MD Expert Report of Steven H. Dikman, MD

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